UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF OKLAHOMA

DOUG INGRAM, et al., Plaintiffs, v. SOLKATRONIC CHEMICAL, INC. and JEFF R. HANNIS, and AIR PRODUCTS AND CHEMICALS, INC., a Delaware corporation, et al. Defendants.	Case No. O4CV287EA(C)

DEFENDANT SOLKATRONIC CHEMICAL, INC.'S
PRIMER ON THE SCIENCE AND MEDICINE OF ARSINE POISONING

Defendant Solkatronic Chemical, Inc. ("Solkatronic"), pursuant to this Court's Order, submits this Primer on the Science and Medicine of Arsine Poisoning.

Respectfully submitted,

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CERTIFICATE OF SERVICE

I hereby certify that I have this 31st day of October, 2005, I electronically transmitted the attached document to the Clerk of the Court using the ECF System for filing and transmittal of a Notice of Electronic Filing to the following ECF registrants:

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INTRODUCTION

192 plaintiffs claim that they suffered permanent injuries caused by exposure to an accidental release of arsine in the Port of Catoosa on July 11, 2001. Twelve of the 192 plaintiffs have been selected as an initial trial group.[1] The plaintiffs procured causation opinions from Drs. Hastings, Gad, and Harrison, identified by the plaintiffs as expert witnesses.

A well established body of scientific and medical literature sets out the signs and symptoms for a diagnosis of arsine poisoning. *The* signature biological effect of arsine is hemolysis. Hemolysis is the destruction of red blood cells. Literature also describes a "classic triad" of symptoms of arsine poisoning. The most prominent of these symptoms is the passing of red urine. Because of its direct, toxic effect on red blood cells, unless certain laboratory results are found, no basis exists to diagnose exposure to arsine.

None of the plaintiffs in this case experienced the classic signs and symptoms of

[1] The parties agreed to proceed with an initial trial group of thirteen plaintiffs. *See* Joint Status Report, filed July 26, 2004 (Docket # 10). Magistrate Cleary eliminated one of the plaintiffs from the initial trial group. *See* Minute Sheet, dated 5/2/05 (Docket #48). The current trial group consists of: Biddle, Cardenas, Castro,

arsine poisoning. None of the plaintiffs experienced the classic triad of symptoms, and none reported having red urine, despite the fact that this objective clinical symptom is almost universally reported in the medical and scientific literature. Moreover, exposure to arsine has truly occurred, many of the plaintiffs were evaluated at local hospitals where they were given blood and urine tests, and none of these tests – which constitute a significant sampling over a period of days – were consistent with the findings required to diagnose exposure to arsine.

Despite all of this, the plaintiffs' experts opine that all (Dr. Hastings) or most (Drs. Gad and Harrison) of the twelve lead plaintiffs were injured by exposure to arsine on July 11, 2001 (or, in some cases, on some other unspecified date(s)). In fact, Dr. Hastings has opined that *virtually every single one of the 192* plaintiffs in this case suffered immediate and permanent injuries from exposure to arsine, even though none showed the recognized signs and symptoms of arsine poisoning, and despite the fact that *permanent* injuries from exposure to arsine are rare and only associated with *severe* cases of arsine poisoning.

In other words, according to the plaintiffs' experts, the plaintiffs in this case are exceptions to everything that is known about arsine poisoning. The plaintiffs somehow experienced arsine poisoning without experiencing the classic signs of such, and they somehow suffered permanent injuries even though such injuries are not medically expected. Either the overwhelming bulk of the scientific and medical literature regarding arsine toxicity is all wrong, or the plaintiffs' experts are.

What's more, the plaintiffs' experts reached their opinions without the most basic

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Guerra, Haggard, Hinton, Ingram, Miller, Patton, Schnitzer, Shavers, and Sumter.

information necessary to establish the possibility (let alone plausibility) of exposure (let alone exposure to an injurious dose). In fact, the plaintiffs' experts reached their causation opinions regardless of and without knowledge of, whether the plaintiffs were inside or outside or upwind or downwind of the release; without knowledge of how far away the plaintiffs were located; without knowledge of how much arsine was released; without attempting to determine the amount and duration of the plaintiffs' exposure (if any); and, perhaps most incredibly, regardless of whether the plaintiffs were present in the Port of Catoosa at the time of the release or, instead, arrived hours or even days later.

The accurate diagnosis of arsine poisoning must be made using an established scientific and medical methodology. Without reliable evidence of exposure and dose, and without any evidence of the established signs and symptoms of arsine poisoning, the plaintiffs' experts clearly did not *follow* a valid scientific methodology to reach causation opinions in this case. Unfortunately, the plaintiffs' experts started with their conclusions and then looked for (and continue to look for) evidence to support their foreordained results. This is not science; it is advocacy.

For all the reasons set forth below, the plaintiffs' experts should be precluded from testifying at trial.

THE SCIENCE AND MEDICINE OF ARSINE POISONING

I. The Biological Effects, Signs and Symptoms of Arsine Poisoning

One of the central tenets of toxicology is that "each chemical agent tends to produce a specific pattern of biological effects that can be used to establish disease causation."[2] The plaintiffs' expert, Dr. Harrison, agrees, referring to these specific biological effects as

[2] Federal Judicial Center, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 403 (20020 ed.).

"markers." *See* Depo. of Dr. Harrison, at 11, ll. 7-9, attached as Ex. 1 ("There is a specific list of signs or symptoms that I would expect from each particular [kind of] chemical exposure.").

The signature biological effect of arsine is that it causes "rapid and fulminant" hemolysis – *i.e.*, the rapid and severe rupturing of red blood cells.[3] Additionally, a well established "triad" of symptoms "ha[s] long been associated with arsine poisonings and stand out as characteristic clinical features."[4] Finally, because arsine has its primary, direct biological effect on red blood cells, a well established set of laboratory results characterizes arsine poisoning.[5]

A. *The* Signature Biological Effect of Arsine is Hemolysis.

Arsine is non-irritating to the nose and lungs, and, when inhaled, it passes easily through the lungs and into the blood stream.[6] There, it quickly destroys red blood cells, causing these cells to rupture and spill their contents into the blood stream. This is known as intravascular hemolysis. Hemolysis is the rupturing or destruction of red blood cells, and "intravascular" hemolysis describes hemolysis that occurs within the bloodstream (as distinguished from hemolysis that may occur outside the bloodstream, for example, during

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[3] Morris Kleinfeld, *Arsine Poisoning*, 22 J. OCCUP. MED. 820, 821 (Dec. 22, 1980) ("The rapid and fulminant hemolysis of red blood cells is a unique and characteristic feature of arsine poisoning."); Bruce Fowler & Joseph Weissberg, *Arsine Poisoning*, 291 NEW ENG. J. MED. 1171, 1171 (Nov. 28, 1974); Agency for Toxic Substances & Disease Registry, *Medical Management Guidelines (MMGs) for Arsine (AsH_3)* 2, http://www.atsdr.cdc.gov/MHMI/mmg169.html [hereinafter *Medical Management Guidelines for Arsine*].

[4] Fowler & Weissberg, *supra* note 3, at 1171.

[5] *Id.*; Martin Caravati, *Arsenic & Arsine Gas*, MEDICAL TOXICOLOGY 1393, 1395 (3d ed. Dart 2003); *Medical Management Guidelines for Arsine*, *supra* note 3, at 2; Dean Carter & John Sullivan, *Intermetallic Semiconductors & Inorganic Hydrides*, MEDICAL TOXICOLOGY OF HAZARDOUS SUBSTANCES 917 (John B. Sullivan et al. eds. 1992).

[6] William Klimecki &Carter, *Arsine Toxicity: Chemical and Mechanistic Implications*, 46 J. TOX. & ENVIRTL. HEALTH 399 (1995); Caravati, s*upra* note ~~6~~5, at 1399.

the collection and processing of blood samples).[7]

The rapid and severe destruction of red blood cells is *the* signature "biological effect" of arsine poisoning,[8] as even the plaintiffs' experts concede.[9] "Acute intravascular hemolysis develops within hours and may continue for up to 96 hours."[10] Hemolysis becomes evident within the first twenty-four hours of the exposure.[11] Even low doses of arsine result in evidence of hemolysis.[12] Severe arsine poisoning can cause renal failure and death, primarily because the kidneys are overwhelmed by the contents (in particular the hemoglobin) of the ruptured red blood cells.[13] "The accepted treatment for acute and severe arsine intoxication is exchange transfusion of the blood and, if renal failure develops, hemodialysis."[14] None of the 192 plaintiffs received any such treatment.

B. Injury Cannot Result from Arsine Without Demonstrable Hemolysis.

Injury from exposure to arsine occurs primarily as a result of – *and in any event does not occur in the absence of* - observable, demonstrable hemolysis. When the plaintiffs'

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[7] *See, e.g.*, University of Virginia Health System, *Hemolytic Anemia*, *available at* http://www.med-ed.virginia.edu/courses/path/innes/rcd/hemo.cfm.

[8] Morris Kleinfeld, *Arsine Poisoning*, 22 JOURNAL OF OCCUPATIONAL MEDICINE 821, 821 (Dec. 22, 1980) ("The rapid and fulminant hemolysis of red blood cells is a unique and characteristic feature of arsine poisoning."); Fowler & Weissberg, *supra* note 3, at 1171.

[9] *See* Depo. of Dr. Harrison, at 35, ll. 6-8, attached as Ex. 1; Depo. of Dr. Gad, at 22, l. 25 – 23, l. 4, attached as Ex. 2; Depo. of Dr. Hastings, at 190, ll. 9-13, attached as Ex. 3.

[10] *Medical Management Guidelines for Arsine*, *supra* note 3, at 5; Center for Disease Control, *Facts About Arsine* 1 (Aug. 29, 2003), *available at* http://www.bt.cdc.gov./agent/arsine/pdf/arsinefactsheet.pdf [hereinafter *CDC's Facts About Arsine*].

[11] *Medical Management Guidelines for Arsine*, *supra* note ~~6~~3, at 7 ("Onset of hemolysis may be delayed for *up to* 24 hours . . . .").

[12] Carter & Sullivan, *supra* note 5, at 918; Klimecki & Carter, *supra* note 6, at 402.

[13] George Jenkins, *Arsine Poisoning*, 2 BRITISH MEDICAL JOURNAL 78 (July 10, 1965). Renal failure only occurs in severe arsine poisoning cases directly results from rapid and severe hemolysis. Thus, the renal failure associated with arsine poisoning occurs soon after the exposure, while the patient is in crisis. Only one plaintiff (Bart Schnitzer) experienced renal failure after the release. Mr. Schnitzer had pre-existing kidney problems which became evident six months after the release, and had no laboratory tests consistent with arsine toxicity. Even Dr. Harrison agrees that Mr. Schnitzer's renal failure was not caused by the arsine release. *See* Depo. of Dr. Harrison, at 155, lns. 4-12, attached as Ex. 1.

[14] Dean Carter, et al, *The Metabolism Of Inorganic Arsenic Oxides, Gallium Arsenide, And Arsine: A Toxicochemical Review*, 193 TOX. & APPLIED PHARM. 309, 311 (2003).-

counsel first contacted one of their experts, Dr. Harrison, they explained to him that "most" of the plaintiffs in this case do not have any evidence that they experienced hemolysis.[15] Accordingly, the plaintiffs' counsel asked Dr. Harrison to consider whether persons can be injured by arsine without experiencing hemolysis.[16]

Dr. Harrison did not answer this question in his written report, but he did at his deposition. Initially, Dr. Harrison explained that exposure to arsine can occur without causing hemolysis (*e.g.*, if the dose is sufficiently low). The following exchange then occurred:-

> Q. And could they hypothetically have health effects without evidence of hemolysis?
>
> A. The individuals could have psychological effects as a result of the exposure, ***but I doubt whether they could have physical effects without evidence of hemolysis***.

See Depo. of Dr. Harrison, at 118, ll. 16-25, attached as Ex. 1 (emphasis added). In other words, even plaintiffs' expert, Dr. Harrison, agrees that although a person could be *exposed* to an amount of arsine insufficient to cause measurable hemolysis, such an exposure will not produce physical injuries. Or, put another way, where injuries are alleged as a result of arsine exposure, there must be evidence of hemolysis.

Plaintiffs' experts are unable to cite any medical or scientific literature to the contrary. *See* Depo. of Dr. Hastings, at 232, ll. 10-25, attached as Ex. 3 ("I don't recall any"); *id.* at 265, l. 23 – 266, l. 8 ("I haven't encountered [any literature] up to this point . . . "); *id*. at 292, l. 12 – 293, l. 7 ("If I've seen [any literature], I can't put my finger on it"); *id*. at 363, l. 17 – 366, l. 17 (he is "not aware of any medical literature where there are delayed

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15 Depo. of Dr. Harrison, at 83, ll. 4-22, attached as Ex. 1.

16 *Id.*

effects following an arsine exposure where there was no hemolysis at all."). Again, Dr. Harrison agrees, stating that he does not "think you'll find an opinion . . . in the literature" that a person can have "arsine exposure sufficient to cause physical symptoms without having a level of hemolysis."[17]

Yet, somehow, plaintiffs' experts opine that ***all*** (Dr. Hastings) or ***most*** (Drs. Gad and Harrison) of the plaintiffs in this case ***did*** suffer physical injuries as a result of exposure to arsine. As is explained below, none of the plaintiffs in this case suffered the classic signs and symptoms of arsine poisoning, including hemolysis, and there is every good reason to doubt (as Dr. Harrison explained) that the plaintiffs were injured.

C. The Classic Triad of Symptoms

The medical and scientific literature consistently describes a classic "triad" of symptoms that "ha[s] long been associated with arsine poisonings and stand out as characteristic clinical features."[18] These three symptoms are (1) abdominal pain; (2) hemoglobinuria (*i.e.*, red urine);[19] and (3) "jaundice" (a yellow or bronze discoloration of the skin).[20] The plaintiffs' experts agree (as they must) that the medical and scientific literature describes this well established triad of symptoms associated with arsine poisoning.[21]

Two of these three signs (red urine and jaundice) are objective findings that are known to be a direct result of the hemolysis caused by arsine poisoning. Frank

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[17] *See* Depo. of Dr. Harrison, p. 178, l. 23 to 179, l. 7, attached as Ex. 1.

[18] Fowler & Weissberg, *supra* note 3, at 1171; Klimecki & Carter, *supra* note 6, at 402.

[19] The medical literature often identifies this symptom as "hematuria." Hematuria is blood in the urine, but the term can refer to whole red blood cells in the urine. Hemoglobinuria is a more precise term because it describes the presence in the urine of hemoglobin from ruptured (not whole) red blood cells. In cases of arsine poisoning, "[u]rinalysis shows large amounts of protein and free hemoglobin usually without intact RBCs." *CDC's Facts About Arsine*, *supra* note 10, at 4.

[20] *Id.*

[21] *See* Depo. of Dr. Hastings, at 172, l. 20 to 171, l. 4, attached as Ex. 3; Depo. of Dr. Harrison, at 170, l. 17-23, attached as Ex. 1; Depo. of Dr. Gad, at 225, ls. 3-10, attached as Ex. 2.

hemoglobinuria (*i.e.*, red urine) is the "tell-tale," objective sign of arsine poisoning. That is, it is the sign of arsine poisoning readily apparent to the patient and treating doctors.[22] "The occurrence of dark-red urine is frequently noticed, usually four to six hours after inhalation of the gas . . . ."[23]

None of the 192 plaintiffs in this case experienced the classic triad of symptoms – a fact that the plaintiffs' experts concede. *See* Depo. of Dr. Harrison, at 169, l. 21 – 170, l. 23, attached as Ex. 1; Depo. of Dr. Gad, at 349. ll. 5-7, attached as Ex. 2; Depo. of Dr. Hastings, at 228, l. 1-9, attached as Ex. 3. ***Not one.*** More specifically, there is no medical evidence in this case that *any* of the 192 plaintiffs had hemoglobinuria (*i.e.*, red urine) or jaundice after the arsine release on July 11, 2001. In fact, there is not even a self-report of these symptoms. In other words, *none* of the plaintiffs have *any* evidence of two of the three symptoms that are characteristic of arsine poisoning. Significantly, these two symptoms are the two symptoms of the triad that could have been (but were not) an objective, clinical finding. Of the twelve lead plaintiffs, only two came close to complaining of abdominal pain, and both of those plaintiffs complained of having this pain *before* the early-afternoon arsine release took place.[24] Thus, *none* of the lead twelve plaintiffs in this case had post-

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[22] *See, e.g.,* Dr. William Banner's Expert Report 2, attached as Ex. 4 (explaining that "[c]linical arsine poisoning presents with . . . a signature injury of hemoglobinuria readily identified as a sudden change in the color of urine. . . ."); *see* expert report from Dr. Pike, at 22, attached as Ex. 5 ("Hemoglobinuria is *sina qua non* of arsine toxicity, and is a specific observation usually present in a plainly visible manner. If hemoglobinuria is not plainly visible, any reported history [of] arsine exposure must be doubted or categorized as not clinically important, e.g. *de minimis*."); Dr. Carter's Expert Report, at 22, attached as Ex. 6.

[23] Fowler & Weissberg, *supra* note 3, at 1171; *accord Medical Management Guidelines for Arsine, supra* note 3, at 3 ("Hemoglobinuria usually occurs within hours.").-

[24] The medical records of one plaintiff, Joshua Hinton, show that he ***woke up*** the morning of July 11, 2001 with "nausea, vomiting, headache, shortness of breath, and also some mild abdominal discomfort, especially after urinating." *See* St. Francis History & Physical Report for Joshua Hinton, at 1, attached as Ex. 7. He and his step-father went to work at the Port of Catoosa but according to his medical records left at 11 a.m. (2 hours before the arsine release) because they felt ill. *Id*. Another plaintiff, Ms. Castro, went to a gastroenterologist on the morning of July 11, 2001 for pre-existing stomach pains. *See* Castro's

release onset of *any* of the classic triad of symptoms associated with arsine.

No medical or scientific literature supports diagnosing arsine poisoning without *any* of the classic triad of symptoms (nor is there any literature that supports basing a diagnosis solely on a subjective report of nonspecific symptoms, like headaches, fatigue, or abdominal pain). The absence of *any* reports of, or clinical evidence of, hemoglobinuria is particularly significant because it is so "frequently" associated with arsine poisoning, "usually" within a few hours of exposure,[25] and is so unlikely to be missed by a person suffering such an effect.

D. Laboratory Results Associated With Arsine Poisoning

The medical literature regarding arsine consistently describes certain lab results that characterize arsine poisoning. These results are all related to the hemolysis that is the signature biological effect of arsine.[26]

1. Hemoglobin Levels Decline below 10 g/dl.

"The most striking and consistent laboratory finding [in cases of arsine poisoning] is anemia of a hemolytic type."[27] Anemia refers to a low red blood cell count or low hemoglobin levels. Hemoglobin is contained in the red blood cells and is "necessary for effective transport of oxygen and carbon dioxide between the lungs and tissues."[28] In cases of arsine poisoning, a low hemoglobin reading or a low hematocrit reading directly reflects

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Regional Medical Laboratory Report, dated 7/11/01, attached as Ex. 8).

[25] *See* Kleinfeld, *supra* note 3, at 820 (noting the case study of a 31-year old truck driver presenting at the hospital following an exposure to arsine with the single symptom of passing dark red urine.). *See also* Luke Yip & Richard Dart, Subchapter on *Arsine Gas* 864 (attached as an exhibit to Dr. Gad's deposition) ("Dark-red discoloration of the urine, hemoglobinuria, or hematuria frequently appears 4 – 12 hours after inhalation of arsine gas.").

[26] *See, e.g.*, Expert Report of Dr. Carter, at 22, attached as Ex. 6 ("Toxicity of arsine is almost exclusively related to hemolysis (no other has been found) . . . .").-

[27] Fowler & Weissberg, *supra* note 3, at 1171; Q. Scott Ringenberg et al, *Hematologic Effects of Heavy Metal Poisoning*, 81 SOUTHERN MEDICAL JOURNAL 1132, 1137 (Sept. 1988) ("Acute hemolytic anemia with hemoglobinemia and hemoglobinuria is the most striking laboratory finding of arsine poisoning."); G.G. Parish et al, *Acute Arsine Poisoning in Two Workers Cleaning a Clogged Drain*, 34 ARCH. ENVIRT'L HEALTH 224, 225 (July/Aug. 1979).

the loss of red blood cells due to hemolysis, and hemoglobin levels are expected to fall below 10 g/dl.[29]

None of the plaintiffs who were tested had hemoglobin readings that were below 10 g/dl. In other words, none of the plaintiffs experienced the "most striking and consistent laboratory finding" in cases of arsine poisoning (just as none of them experienced the classic triad of symptoms). In fact, of the twelve lead plaintiffs in this case, there are no reported laboratory values of hemoglobin below the normal range, let alone below 10 g/dl.[30] To the contrary, several plaintiffs had normal or even "high" normal readings.[31]

2. Plasma Free Hemoglobin Levels "Often" Exceed 2000 mg/dl and May Be Much Higher.

When hemolysis occurs (*i.e.*, when the red blood cells are destroyed), hemoglobin contained in the red blood cells is released into the plasma. This results in an increase in a person's "plasma hemoglobin" or "plasma free hemoglobin."

The medical and scientific literature regarding arsine poisoning explains that plasma free hemoglobin levels "often" rise above 2000 mg/dl (milligrams per deciliter), [32] with

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[28] CASARETT & DOULL'S TOXICOLOGY: THE BASIC SCIENCE OF POISONS 818 (6th ed. 2001).

[29] *See, e.g.*, Klimecki & Carter, *supra* note 6, at 403; Dean Carter, et al., *Sequence of Toxic Events in Arsine-Induced Hemolysis In Vitro: Implications for the Mechanism of Toxicity in Human Erythrocytes*, 38 FUND. & APPLIED TOX. 123 (1997); Fowler & Weissberg, *supra* note 3, at -1171.

[30] The plaintiffs' counsel provided each of their experts with a chart indicating the locations of the plaintiffs who had "high" rather than "low" - hemoglobin levels. *See* Plaintiffs' High Hemoglobin Chart, attached as Ex. 9. Four of the twelve lead plaintiffs are listed on the chart. As explained in the body of this brief, these plaintiffs' high hemoglobin readings contradict a finding of arsine exposure.

[31] The reference or normal range for hemoglobin is between 12.3 and 17.0 grams per deciliter. Plaintiffs Cardenas (16.7 g/dl), Sumter (16.7 g/dl), Hinton (15.6 g/dl) and Ingram (15.5 g/dl) all had normal/high hemoglobin readings. *See* Chart of Clinical Findings for Twelve Plaintiffs, attached as Ex. 10. The lab results for the remaining plaintiffs who were tested were all within in the normal or reference range: Castro (12.7 g/dl taken on 7/14), Miller (13.3 g/dl), Patton 13.9 g/dl), and Biddle (15.3 g/dl). *Id.* Plaintiffs Guerra, Haggard, Schnitzer and Shavers did not go to the hospital after the arsine release.

[32] *See* Klimecki & Carter, *supra* note 6, at 403 (18,500 mg/dl); Carter & Sullivan, *supra* note 5, at 918; Fowler & Weissberg, *supra* note 3, at 1171 ("Plasma hemoglobin values of greater than 2 g per 100 ml [or 2000 mg/dl] are reported.").

reports as high as 18,500 mg/dl.[33] Dr. Harrison authored a text section that discussed arsine. Dr. Harrison stated that "[a] history of arsine exposure and a plasma hemoglobin of greater than 1.5% confirms the diagnosis of arsine poisoning."[34] A plasma free hemoglobin reading of "1.5%" is the same as 1500 mg/dl.[35]

None of the plaintiffs had plasma free hemoglobin test results that were anywhere near the 2000 mg/dl (up to 18,500 mg/dl) widely cited in the literature as being characteristic finding of arsine poisoning. *None* of the plaintiffs in this case had a plasma free hemoglobin reading that was even *one-tenth* the level (1500 mg/dl) that plaintiffs' own expert, Dr. Harrison, identified as "confirm[ing] the diagnosis of arsine poisoning."[36] Despite these facts and the established literature on this issue (including the literature of the plaintiffs' own expert, Dr. Harrison), the plaintiffs' experts have relied on *de minimis* increases in plasma free hemoglobin as evidence of arsine poisoning sufficient to cause acute and permanent injuries. The plaintiffs' experts have no scientific or medical basis to do this, and they themselves are unable to cite any support for their theories. This issue is discussed in greater detail below in Part G.

3. Haptoglobin Levels Decline Quickly and Are Frequently Absent.

In ordinary, healthy individuals, red blood cells age and rupture. When that happens in the ordinary course, the released hemoglobin reacts with haptoglobin normally circulating

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[33] Klimecki & Carter, *supra* note 6, at 403; Carter, *supra* note 29, at 123; Fowler & Weissberg, *supra* note 3, at 1171; Shannon Winski et al, *Sequence of Toxic Effects in Arsine-Induced Hemolysis in Vitro: Implications for the Mechanism of Toxicity in Human Erythrocytes*, 38 FUND. & APP. TOXIC. 123, 123 (1997); Carter, *supra* note 14, at 193 ("[P]lasma hemoglobin levels often rise to greater than 2 grams per deciliter (or 2000 mg per ml)."). The medical and scientific literature often reports the expected plasma free hemoglobin level as 2 grams per deciliter (2 g/dl) or 2 grams per 100 milliliters (2 g/100ml), both of which are equivalent to 2000 mg/dl. Because the lab results in this case are expressed in milligrams per deciliter (mg/dl), this Primer uses that unit of measurement and the values reported in the literature have been converted to mg/dl for ease of comparison.

[34] Robert Harrison, *Chemicals & Gases*, 27 OCCUP. & ENVTL. MED. 917, 974 (Dec. 2000).

in the bloodstream and is cleared from the body. When arsine exposure causes hemolysis, much more hemoglobin is released into the blood plasma than normal, causing haptoglobin to decline quickly. The decline in haptoglobin is, therefore, a direct consequence of the sudden and significant increase in plasma free hemoglobin in the blood.

In cases of arsine poisoning, "[h]aptoglobin levels decline ***rapidly***,"[37] and are "***frequently absent***," (*i.e.*, drop to zero).[38] If haptoglobin levels have not declined to zero, then any hemolysis that may have occurred has not been sufficient to overwhelm the body's normal ability to process the rupturing of red blood cells. In other words, if haptoglobin levels have not gone to zero, then there is no reason to believe that there can be injury from an alleged exposure to arsine.

None of the plaintiffs in this case had a haptoglobin test result that went below normal, much less to zero.[39] All haptoglobin results were within the laboratory normal range.[40] In fact, several of the twelve lead plaintiffs had "high" normal levels of haptoglobin.[41] The absencel of below normal haptoglobin is significant because it indicates that, even if there was exposure to arsine and some resulting hemolysis (and there is no evidence to this effect), the body's natural mechanism for dealing with hemolysis in a harmless manner was not overwhelmed. The plaintiffs' haptoglobin level dropping below normal or to zero is demonstrable of absolutely no injury from arsine.

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35 *See* Depo. of Dr. Harrison, at 353, ll. 20-22, attached as Ex. 1.

36 Robert Harrison, *Chemicals & Gases*, 27 OCCUP. & ENVTL. MED. 917, 974 (Dec. 2000).

37 *See Medical Management Guidelines for Arsine*, supra note 3, at 3.

38 ENVNT'L & OCCUP. HEALTH 392 (William Rom, ed. 1983) ("As hemoglobin binds with haptoglobin, the concentration of the latter declines and is frequently absent."); Marchand et al, *The Predictive Value Of Serum Haptoglobin In Hemolytic Disease*, J. AMER. MED. ASS'N 1909-11 (1980).

39 *See* Depo. of Dr. Hastings, 198, ll. 11-18, attached as Ex. 3 (agreeing that he is not aware of any plaintiff with a haptoglobin level outside of normal). *See also* Chart of Clinical Findings for Twelve Plaintiffs, attached as Ex. 10.

40 *See* Chart of Clinical Findings for Twelve People, attached as Ex. 10.

4. Urinalysis Is Positive for Hemoglobinuria.

As already noted, hemoglobinuria is the "tell tale" sign of arsine poisoning. A urine dip stick test can confirm or rule out the presence of "blood" in the urine. A negative test rules out hemoglobinuria. A positive dip-stick test, however, confirms only the presence of "blood" in the urine, but it does not distinguish between hematuria (whole red blood cells in the urine, which is not caused by arsine exposure) and hemoglobinuria (which is caused by arsine exposure).

To determine if a positive dip-stick test is hematuria or hemoglobinuria, a sample is viewed under a microscope to check for the presence of whole red blood cells.[42] If whole red blood cells are present, hematuria (not hemoglobinuria) accounts for the positive urine dip stick test. If the urine dip stick test is positive but the microscopic sample is negative for whole red blood cells, hemoglobinuria is confirmed.[43] Hemoglobinuria - not hematuria - is the laboratory result if there is an arsine injury.[44]

No medical evidence in this case shows that *any* of the twelve plaintiffs had hemoglobinuria after the arsine release on July 11, 2001.[45] In fact, there is not even a self-report of this symptom.[46] Although some of the plaintiffs had positive dip-stick tests for blood in their urine, <u>*each*</u> of these plaintiffs were subsequently found by microscopic

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41 *Id.*

42 LABORATORY TEST HANDBOOK 216-17 & 908 (2ND ED. 1990). *See also* Depo. of Dr. Harrison, at 163, l. 7 – 164, l. 20, attached as Ex. 1.

43 Kleinfeld, *supra* note 3, at 821("[T]he finding of hemoglobinuria rather than intact red blood cells should have alerted the physicians earlier to the correct diagnosis.").

44 The plaintiffs' counsel apparently misunderstood the significance of a positive urine dipstick test as they supplied their experts with a chart listing the plaintiffs with high hematuria – not high hemoglobinuria. *See* Plaintiffs' High Hematuria Chart, attached as Ex. 9.

45 *See* Chart of Clinical Findings for Twelve Plaintiffs, attached as Ex. 10.

46 One plaintiff, Mr. Hinton, claims that he had dark urine two years after the incident. However, Mr. Hinton's medical records from the day of the incident show that his urinalysis showed clear urine and further show that he was not present at the time of the incident.

examination to have whole red blood cells in their urine.[47] In other words, these plaintiffs had hematuria (which is not associated with arsine poisoning), but *none* of them had hemoglobinuria (which is).[48]

5. Nonspecific Symptoms Are Not Diagnostic of Arsine Poisoning, Especially Without Evidence of Hemolysis.

Although plaintiffs' experts argue that there is some evidence of hemolysis, namely the slight elevations in plasma free hemoglobin in a few of the plaintiffs (which are discussed below), they agree (as plaintiffs' counsel apparently does as well) that in *most* cases there is no evidence of hemolysis whatsoever. As a result, in many cases, the plaintiffs' experts rely primarily on alleged nonspecific symptoms (*i.e.*, headache, malaise, fatigue, shortness of breath, nausea) to support their opinion that the plaintiffs suffered arsine poisoning on July 11, 2001. These nonspecific symptoms are not the "biological effects" or "markers" used to establish causation in cases of suspected arsine poisoning, and they cannot be used to diagnose arsine poisoning.

To begin with, "[t]hese types of symptoms are part of human experience and can be triggered by a host of medical and psychological conditions,"[49] including dehydration from heat exposure which a number of persons treated on July 11, 2001 experienced.[50] More

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[47] Two other plaintiffs (Ms. Haggard and Mr. Cardenas) initially tested positive for blood in the urine using the dipstick test. However, the follow up test for Mr. Cardenas and Ms. Haggard showed that neither had plasma free hemoglobin in their urine. *See* Depo. of Harrison, at 262, l. 24 – 263, l. 5, attached as Ex. 1. Dr. Hastings, agrees that Ms. Haggard's "diagnostic tests did not reveal evidence of significant anemia outside of the normal levels expected at pregnancy." *See* Report of Richard Hastings, D.O. on Plaintiff, Theresa Haggard, at 2, attached as Ex. 192.

[48] *See* Depo. of Dr. Harrison, at 209, l. 19 – 210, l. 4, attached as Ex. 1. Dr. Harrison only seemed to realize during his deposition that the positive dip-stick tests were subsequently determined to be negative for hemoglobinuria. *Id*. at 164, l. 21 – 165, l. 17.

[49] REFERENCE MANUAL, *supra* note 2, at 426.

[50] One of the plaintiffs, Mr. Ingram, explained that he was immediately given an IV to re-store the hydration in his body. He estimates that he lost 10 to 15 lbs. from standing in the sun. *See* Depo. of Douglas Ingram, at 116, l. 22 – 117, l. 23, attached as Ex. 143. Another of the twelve plaintiffs, Allen Miller, was diagnosed with 1st degree sunburn when he arrived at hospital. *See* Medical Records for Miller

importantly, most of the nonspecific symptoms associated with arsine poisoning are known to be a direct result of the hemolysis that arsine causes. For example, the headaches, fatigue, and shortness of breath associated with arsine poisoning are all secondary symptoms of hemolysis.[51]

What this means is that the plaintiffs and their experts cannot reasonably rely on subjective reports of headaches, shortness of breath, and fatigue to support a diagnosis of arsine poisoning when there are no concurrent signs and symptoms of hemolysis. Stated another way, where laboratory tests were negative for hemolysis (which is true here in all cases in which laboratory tests were taken), nonspecific symptoms ***must*** have had another cause (*e.g.*, the possibility that the plaintiffs were suffering from heat exposure and dehydration as a result of being evacuated to outdoor locations in the extreme temperatures on July 11, 2001).

On this issue, the plaintiffs and their experts simply cannot have their cake and eat it too. The plaintiffs want to rely on contemporaneously occurring, nonspecific symptoms as evidence of arsine poisoning while simultaneously arguing that the absence of evidence of hemolysis is not problematic because such hemolysis may have been delayed. Without hemolysis, the plaintiffs cannot reasonably claim that their nonspecific symptoms were caused by arsine because, in cases of arsine poisoning, these nonspecific symptoms are caused by hemolysis.[52]

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from Tulsa Regional Medical Center, at 2, attached as Ex. 14. Several others acknowledged that they stood outside in temperatures of 105 degrees or more. *See, e.g.*, Depo. of Charles Biddle, at 46, l. 2-19, attached as Ex. 15.

[51] "After arsine enters the bloodstream, it damages the red blood cells and leads to symptoms as a direct result of this damage." *CDC's Facts About Arsine*, *supra* note 10, at 1. *See also* Nora Goldschlager & Neal Benowitz, *Arsenic and Arsine*, CLINICAL MANAGEMENT OF POISONING AND DRUG OVERDOSE 788 (3rd ed. Haddad et al. 1998).

[52] *See* Depo. of Dr. Pike, at 112, l. 19 – 113, l. 13, attached as Ex. 16.

E. Long-Term Health Effects Are Rare and Not Expected Without Evidence of a Severe Exposure.

Most persons recover fully from acute (*i.e.*, non-chronic), non-lethal arsine poisoning. "Most people do not develop long-term effects from a single, small exposure to arsine. In rare cases, permanent kidney damage or nerve damage has developed after a severe exposure."[53] Permanent injuries from acute exposures are rare and only follow severe poisoning, which is characterized by massive hemolysis requiring significant medical intervention (*i.e.*, exchange transfusions and dialysis).[54] Amazingly, the plaintiffs (and two of their experts, Dr. Hastings and Dr. Gad) claim that each of the plaintiffs suffered these long-term effects from arsine exposure ***without*** experiencing the root cause of those effects, *i.e.*, hemolysis. The plaintiffs offer no scientific or medical support for such a jump in logic, and the scientific literature flatly belies their claims.

F. The Plaintiffs Made No Effort to Estimate Dose, Despite Acknowledging that Such Information Was "Important" and "Relatively Easy" to Calculate.

1. Reliable, Scientific Dose Information Is Required as a Matter of Law.

It is a fundamental tenet of toxicology – and toxic-tort case law – that the "dose makes the poison." *See, e.g.*, *Mancuso v. Con. Edison Co. of New York*, 967 F. Supp. 1437 (S.D.N.Y.1997); REFERENCE MANUAL, *supra* note 2, at 403. This tenet recognizes that any substance can be injurious in sufficiently high doses (including, for example, water) and, conversely, that any substance can be non-injurious in sufficiently low doses (including arsine). Whether, and to what extent, an injury occurs depends on the dose. *Id*.

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53 *See Medical Management Guidelines for Arsine, supra* note 3, at 9.

54 *Id*.

The question in a toxic tort case like this one, then, is not whether the plaintiffs were *exposed* to arsine. Rather, the question is whether the plaintiffs were exposed to an *injurious* dose of arsine – or, put another way, whether they suffered arsine poisoning or arsine intoxication (not just arsine exposure). *See, e.g., McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1242 (11th Cir. 2005) (What is required is "not simply proof of exposure to the substance, but proof of enough exposure to cause the plaintiff's specific illness"). To this end, the law is clear in the Tenth Circuit (and others) that, to meet their burden of proof in this case, the plaintiffs and their experts are ***required*** to present reliable evidence that the plaintiffs were exposed to a dose of arsine sufficient to cause injury. *See Mitchell v. Gencorp Inc.*, 165 F.3d 778 (10th Cir. 1999). The Court in *Mitchell* made the rules that govern this case very clear:

> "Scientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiffs' burden in a toxic tort case." . . . .[A] plaintiff must prove level of the exposure using techniques subject to objective, independent validation in the scientific community. . . . At a minimum, the expert testimony should include a description of the method used to arrive at the level of exposure and scientific data supporting the determination.

Id. (quoting *Allen v. Pennsylvania Eng. Corp.*, 102 F.3d 194, 199 (5th Cir. 1996)).

Consistent with these requirements, establishing (1) the dose required to cause injury (general causation), and (2) that the plaintiff at issue was exposed to such a dose (specific causation), are established components of accepted methodologies for assessing injury in a toxic tort case. *See, e.g., Mancuso*, 967 F. Supp. at 1441. And, "[c]ourts have held that even if an expert seeking to testify is not a toxicologist, he must employ principles and methods of toxicology if he is to give an opinion on an issue relating to that specialty." *Id.*

As explained below, none of the causation opinions of the plaintiffs' experts'

opinions include a description of the method used to arrive at the level of exposure and scientific data supporting the determination, as is required by *Mitchell, supra*. In fact, the plaintiffs' experts have offered their causation opinions, not only without obtaining scientific information regarding dose, but also without even obtaining the most basic facts necessary to establish a reasonable possibility of exposure to any dose. These failures are fatal as a matter of law. *Id.*

2. The Plaintiffs' Experts Have No Evidence of "Dose."

Generally speaking, dose is a function of the amount and the duration of the exposure. In a case like this, involving the release of a toxic gas, the dose to which any particular plaintiff was exposed would be measured by the airborne concentration of the gas as it reached and passed by the plaintiff and the length of time that the plaintiff was exposed to the gas. Although there is often no direct evidence of such information (*i.e.*, there are rarely monitors at the plaintiffs' locations that measured the actual concentrations and the length of exposure at the time of the event), there are well established, scientifically valid means of estimating this information.-

Such an estimate is commonly referred to as a "dispersion analysis" or "dose reconstruction." A dispersion analysis would model the likely plume of the release and the so-called "zone of danger" (*i.e.*, the areas in which it is likely that the concentration and duration of exposure were likely to exceed harmful levels) by taking into account such factors as the amount and duration of the release and the wind and weather conditions at the time.

As is explained in the Federal Judicial Center's Reference Manual on Scientific Evidence:

> Evidence of exposure is essential in determining the effects of harmful substances. . . . [W]hen direct measures cannot be made, exposure can be measured by mathematical modeling, in which one uses a variety of physical factors to estimate the transport of the pollutant from the source to the receptor. For example, mathematical models take into account such factors as wind variations to allow calculation of the transport of radioactive iodine from a federal atomic research facility to nearby residential areas.

REFERENCE MANUAL, *supra* note 2 (*citing In re Three Mile Island Litig. Consol. Proceedings*, 927 F. Supp. 834, 870 (M.D. Penn. 1996); *Mitchell v. Gencorp Inc.*, 165 F.3d 778, 781 (10th Cir. 1999) ("[A] plaintiff must prove level of the exposure using techniques subject to objective, independent validation in the scientific community."); *Wright v. Williamette Indus. Inc.*, 91 F.3d 1105, 1107 (8th Cir. 1996); *Valentine v. Chlor Alkali Co.*, 921 F. Supp. 666, 678 (D. Nev. 1996)). A similar description was given to dispersion modeling in an article in Dr. Gad's files. That article describes how to model a toxic gas release and, coincidentally enough, uses an arsine release as the hypothetical:

> Dispersion models are employed to simulate the airborne concentrations of arsine downwind that could potentially occur following such releases. The objective of dispersion modeling is to provide a conservative estimate of the zone of vulnerability for a given accidental release as a function of certain meteorological conditions. The zone of vulnerability is determined as the maximum width and distance downwind that a specified level of concern (*i.e.*, an airborne concentration) of arsine may potentially be exceeded.

John Lowe et al, *Assessing Community Risk from the Sudden Release of a Toxic Gas* 451, 454.

The plaintiffs themselves have acknowledged that dose information is "important" and that it is "relatively easy" to calculate. In a December 23, 2004, letter to Dr. Harrison, the plaintiffs' counsel explained that "it would appear to be important for us to attempt to demonstrate the highest possible degree of certainty that our plaintiffs were exposed to a

sufficiently high level of arsine to support our physician's differential diagnosis."[55] The plaintiffs' counsel went on to explain that "arsine, when inhaled, can cause injury in concentrations as low as a few parts per million," and that, although "there is no way to know the exact dispersion of the arsine in this case, it is a relatively easy calculation to determine the size of the area in which an injurious concentration could be dispersed."[56] Dr. Harrison was specifically "authorized" to "consult with another expert to obtain expert advice on the likely zone of danger created by the arsine release." *See* Letter from Keith Ward to Dr. Harrison 3 (12/22/04), attached as Ex. 17.

The incident in question occurred over four years ago, and despite the legal mandate in *Mitchell*, the plaintiffs and their experts failed to obtain any dose information that would demonstrate the plaintiffs were exposed to harmful levels of arsine. None of the plaintiffs' experts know how much arsine, if any, may have survived the initial explosion.[57] None know how much arsine, if any, left the Solkatronic property line and went onto the property of its neighbors on July 11, 2001.[58] The plaintiffs' experts cannot even say how much arsine, if any, the twelve plaintiffs in the initial group could have been exposed to or the duration they were exposed to it.[59] *See* Depo. of Dr. Gad, at 136, ll. 22-25, ("Whether any arsine survived this reaction is unknown.").

The plaintiffs' experts reached their causation opinions in this case regardless of, and

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55 *See* Letter from Keith Ward to Dr. Harrison, dated 12/22/04, attached as Ex. 197.

56 *Id.*

57 *See* Depo. of Dr. Hastings, 286, l. 1 – 17, attached as Ex. 3; Depo. of Dr. Gad, at 113, lns. 17-20, attached as Ex. 2. As Dr. Harrison testified, "I do not know. I don't know what happened over a period of hours or even minutes to hour to weeks to months." *See* Depo. of Dr. Harrison, at 148, ll. 1-10, attached as Ex. 1.

58 *See* Depo. of Dr. Hastings, at 286, l. 1 – 17, attached as Ex. 3; Depo. of Dr. Gad, at 110, ll. 13-14, attached as Ex. 2; Depo. of Dr. Harrison, at 148, l. 1- p. 149, l. 1, attached as Ex. 1.

59 *See* Depo. of Dr. Hastings, 286, l. 1 – 17, attached as Ex. 3. As Dr. Gad testified, since there no evidence of dose, "[t]here's no way to do a dose response." *See* Depo. of Dr. Gad, at p. 110, lns 13-14,

without knowledge of, whether the plaintiffs were inside or outside or upwind or downwind of the release;[60] regardless of, and without knowledge of, how far away from the release the plaintiffs were located; without knowledge of how much arsine was released;[61] without attempting to determine the amount and duration of the plaintiffs' exposure (if any); and, perhaps most incredibly, regardless of whether the plaintiffs were even present anywhere in the Port of Catoosa at the time of the release or, instead, arrived hours or even days later.[62] Lack of this required information is fatal to plaintiffs' experts' opinions.

G. The Plaintiffs' Experts' Rely on *De Minimus* Elevations of Plasma Free Hemoglobin While Ignoring Other More Likely Causes of the Elevations.

The plaintiffs prepared a map identifying 19 persons who (allegedly)[63] had plasma free hemoglobin readings above the laboratory reported normal range of 0.0 – 10.4 mg/dl.

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attached as Ex. 2.

[60] *See* Depo. of Dr. Hastings, at 296, l. 13 – 297, l. 19, attached as Ex. 3 ("I don't know. I wasn't there."); Depo. of Dr. Harrison, at 138, ll. 5 – 21, attached as Ex. 1 ("I couldn't tell you. That's beyond my expertise."); Depo. of Dr. Gad, at 42, ll. 21 – 24, attached as Ex. 2.

[61] *See* Depo. of Dr. Hastings, at 111, l. 13 – 112, l. 5, attached as Ex. 3; Depo. of Dr. Harrison, at 147, l. 22 – 148, l. 1, attached as Ex. 1; Depo. of Dr. Gad, at 110, ll. 13-14, attached as Ex. 2.

[62] *See* Depo. of Dr. Hastings, at 111, l. 13 – 112, l. 5, attached as Ex. 3; Depo. of Dr. Harrison, at 147, l. 22 148, l. 1, attached as Ex. 1; Depo. of Dr. Gad, at 110, ll. 13-14, attached as Ex. 2. Dr. Gad asked the Plaintiffs' attorneys for information on the zone of vulnerability (Depo. of Dr. Gad, at 41, lns. 21 - 42, ln. 7) the attorneys never provided the information and the "zone of vulnerability" was never defined. *Id*. at 45, lns. 17-23. The information he did receive - charts or maps prepared by Plaintiffs' counsel showing the location of individuals (*Id*. at p. 42, ln. 5-7) had no scale to determine distances. *Id*. at 43, lns. 12-15. Rather, Dr. Gad makes assumptions as to how far people and buildings are from each other. *Id*. at p. 43, lns. 16-24. And he never visited the Port of Catoosa. *Id*. at p. 237, lns. 11-16 ("I will need to actually see the facility."). Dr. Harrison could not say which way the wind was blowing from the meteorological documents he was provided. *See* Depo. of Dr. Harrison, at 138, ll. 16-21, attached 1as Ex. 1 ("That's beyond my expertise.").–

[63] Two of the persons listed on the plaintiffs' map are not actually plaintiffs in this case (Francisco Aguirre and Dirk Zimmerman). The plaintiffs have not provided Solkatronic with any information regarding Francisco Aguirre, who was never a plaintiff in this case. Zimmerman's medical records were

Although the plaintiffs were at least right that plasma free hemoglobin readings should go up in cases of arsine poisoning, this map is only slightly more useful than the plaintiffs' maps of plaintiffs with *high* hemoglobin (rather than low) and hematuria (rather than hemoglobinuria). *See* notes 30 & 31, *supra*.

The "elevated" plasma free hemoglobin readings identified on the plaintiffs' map range from 10.5 mg/dl[64] to 111 mg/dl.[65] Slight elevated readings such as these are nowhere near the diagnostic level of 1500 mg/dl as set by plaintiffs' expert Dr. Harrison, nor are they anywhere near the levels reported in the medical and scientific literature (*i.e.*, "often" above 2000 mg/dl and as high as 18,500 mg/dl). In fact, only two of the 19 plaintiffs identified on the plaintiffs' map had readings above 50 mg/dl, and eight of the 19 had readings below 20 mg/dl.[66] In other words, although these readings were slightly "elevated" above normal, *they range from roughly eighteen to 200 times lower than the low end of the plasma free hemoglobin readings that are often exceeded in reported cases of arsine poisoning* (*i.e.*, >2000 mg/dl).

Despite this, the plaintiffs' experts have attempted[67] to rely on these so-called

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produced because, at one time, he was a plaintiff in this action.

[64] To even suggest that a reading of 10.5 mg/dl is in any way meaningful – compared to an upper normal range of 10.4 mg/dl and a diagnostic level of 1500 mg/dl for arsine poisoning (as set by plaintiffs' own expert) – is a bit absurd. This is a bit like arguing that a person with a temperature of 98.6001 should be included on a list of persons with a fever.

[65] *See* Plaintiffs' Map of Plasma Free Hemoglobin Readings, attached as Ex. 18.

[66] *See* Chart of Plasma Free Hemoglobin Readings for 19 Plaintiffs, attached as Ex. 11 compare with Dr. Harrison's Chart, attached as Ex. 19.

[67] Dr. Harrison clearly misunderstood the plasma free hemoglobin readings for the plaintiffs. Dr. Harrison testified that "there were many of these individuals that had elevated plasma free hemoglobins. The highest recorded among these individuals . . . was Mr. Sumter . . . [with] a plasma hemoglobin of 111%." Id. at 1698, ll. 4-9, attached as Ex. 1. As *erroneously* reported by Dr. Harrison, the plasma free hemoglobin readings of the five plaintiffs with elevated readings were more than *1,000 to* ***10,000*** times above normal and 7 to 74 times *higher* than the level that Dr. Harrison wrote "confirms the diagnosis of arsine poisoning." However, when the units of measurement are properly understood, the plaintiffs' plasma free hemoglobin readings were only 1.01 to 10 times normal and *13 to 142 times below* the level that "confirms the diagnosis of arsine poisoning." Clearly, the units of measurement are important. Like Dr. Harrison, Dr. Gad did not even give the readings in his expert report, and belatedly looked at these numbers

"elevated" plasma free hemoglobin readings as evidence of arsine poisoning. But, there is no scientific or medical support for doing so. To the contrary, as Solkatronic's experts explain, these readings are not clinically significant,[68] and the plaintiffs' experts have not been able to cite any medical literature to support their claim that these minor elevations of plasma free hemoglobin support a diagnosis of arsine-induced injury.[69]

Moreover, there are substantial, self-evident reasons to doubt that these readings have any significance at all. Three of the 19 plaintiffs with elevated readings were not even present at the time of the release,[70] and five more of the 19 plaintiffs were *away* from the direction of the prevailing wind (in some cases quite far away).[71] In other words, there is every reason to believe that many of these people could not possibly have even been *exposed* to arsine on July 11, 2001. Given that fact, there is clearly no reason to believe that these slight elevations in plasma free hemoglobin are related to arsine.

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only during his deposition. *See* Depo. of Dr. Gad, at 209, l. 5 – 210, l. 16, attached as Ex. 2.

[68] *See, e.g.*, Depo. of Dr. Pike, at 96, l. 19- 97, l. 4, attached as Ex. 146; Depo. of Dr. Banner, at 119, ll. 10 – 17, attached as Ex. 20.

[69] *See* Depo. of Dr. Harrison, at 309, ll. 15-23, attached as Ex. 1 ("I am not aware, as I sit here today, of any literature that answers that question."); Depo. of Dr. Gad, at 188, ll. 4-15, attached as Ex. 2 (Dr. Gad initially pointed to two sources but later admitted that neither supported this opinion); Depo. of Dr. Hastings, at 461, l. 22 – 462, l. 15, attached as Ex. 3 (Dr. Hastings does not respond to the question directly but points to an article written by Dr. Dean Carter in which low to moderate hemolysis – as opposed to plasma free hemoglobin levels – are referenced).

[70] Plaintiffs Hinton, Harper and King were not present at the Port of Catoosa at the time of the incident. Mr. Hinton left with his step-father at 11 am (2 hours before the incident) because they woke up feeling poorly that morning. *See* Medical Records for Joshua Hinton, at 2, attached as Ex. 7. Plaintiff Harper works the night shift and did not arrive at the Port of Catoosa until 8:30 p.m. – 7 ½ hours after the incident. *See* Dr. Hastings' Expert Report on Jeremy Harper, at 2, attached as Ex. 21. Plaintiff King told Dr. Hastings that he was at lunch at the time of the incident but came back to help with the evacuation. *See* Dr. Hastings' Expert Report on Eugene King, attached as Ex. 22.

[71] Plaintiff Thrasher (18 mg/dl) was located to the southeast - in the opposite direction of the prevailing winds. Plaintiff Hessman (13 mg/dl) was far away to the east. Plaintiff Linleys (13.4 mg/dl) and Coatny (45 mg/dl) were located relatively close to Solkatronic's facility, but to the east (*i.e.*, against the prevailing winds). Plaintiffs' map includes a Francisco Aguirre, but Mr. Aguirre is not a plaintiff in this case, and Solkatronic has no information regarding Mr. Aguirre. In any event, Mr. Aguirre was located at Erlanger Tubular, Corp. (where some other plaintiffs were located), one mile to the northeast. Plaintiffs' experts have offered no scientific explanations as to how arsine (in any concentration) could have reached these plaintiffs, let alone how such minor elevations could be clinically significant. *See* Plaintiffs' Map of Plaintiffs with High Plasma Free Hemoglobin, attached as Ex. 18.

1. The Plaintiffs' Experts Disregard Other Possible Causes of the Plantiffs' *De Minimus* Plasma Free Hemoglobin Levels.

There are "many" other possible causes of hemolysis (and, therefore, slightly elevated plasma free hemoglobin readings). The plaintiffs' experts acknowledge this fact,[72] but failed to consider these other explanations, notwithstanding the fact that there is virtually indisputable evidence (just noted above) to doubt that these readings are related to arsine. A few points are worth noting regarding other possible causes of hemolysis and, consequently, other possible causes of elevated plasma free hemoglobin.

First, there are well known causes of false high plasma free hemoglobin readings, which the plaintiffs' experts did not consider or exclude. (These false readings are sometimes referred to as "artifacts."). One type of false high reading can occur when red blood cells rupture during the collection and/or processing of blood samples.[73] For example, if blood is drawn too quickly and/or drawn through a narrow needle, some hemolysis may occur. The hemolysis expected from such mechanical causes is relatively small compared with the significant hemolysis expected in cases of arsine poisoning.[74] This is not "intravascular" hemolysis, but is a well known and not infrequent phenomenon that can lead to an elevated plasma free hemoglobin reading.[75]

These things happened in this case, and that the physicians who actually treated these

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[72] As Dr. Hastings testified, "there's a great number of things that can produce free plasma hemoglobin elevation, various types of - - there's all kinds of hemolytic processes out there." *See* Depo. of Dr. Hastings, at 197, ll.14-22, attached as Ex. 3.

[73] In one study, 13.7% of the patients whose blood was taken by IV catheter were hemolytic and 3.8% of the patients whose blood was taken by venipuncture were hemolytic. *See* Colleen Kennedy et al., *A Comparison of Hemolysis Rates Using Intravenous Catheters Versus Venipuncture Tubes for Obtaining Blood Samples*, 22 J. EMER. NURSING 566 (1996). *See also* Depo. of Dr. Hastings, at 201, l. 10 – 202, 18, attached as Ex. 3.

[74] *See* Depo. of Dr. Hastings, at 199, l. 19 – 207, l. 11, attached as Ex. 3, wherein Dr. Hastings listed potential causes of biochemical evidence of ruptured red blood cells including drawing the blood sample itself, immune disorders, certain medications, march hemoglobinuria, and insect bites.

[75] *See* Depo. of Dr. Banner, at 121, l. 22 – 122, l. 4, attached as Ex. 15 ("And, you know, knowing

patients in July 2001 (doctors who were not hired to give opinions in this case) did not consider the "elevated" readings relied upon by the plaintiffs' experts to be evidence of arsine-induced hemolysis. For example, one treating doctor specifically noted that the elevated readings seen at St. John Medical Center were thought "on most of these folks" to be "due to hemolysis from blood drawn."[76] *Eleven* of the 19 plaintiffs on the plaintiffs' map had their blood work done at St. John Medical Center.

2. Even the Highest Plasma Free Hemoglobin Reading – for Plaintiff Joe Sumter – Was Attributed to Other Causes by the Medical Personnel Who Treated Him After the Incident.

The plasma free hemoglobin reading of Joe Sumter (111 mg/dl) is worth noting because the plaintiffs and their experts have trumpeted this reading – the ***highest*** of anyone in this case – as clear evidence of arsine-induced hemolysis. But Sumter's blood sample was clearly labeled by the lab as being "lipemic" (*i.e.*, fatty and susceptible to a false positive).[77] False high readings can also occur when blood samples are "cloudy." This is because plasma free hemoglobin readings are measured by a light refraction test. As a result, a false high reading can occur, for example, when there is an excessive amount of fat in a blood sample (*e.g.*, from the recent consumption of fatty foods).[78]

What's more, his treating physician (who was aware that there had been an arsine release and was alerted to look for signs of hemolysis) stated in his discharge notes that Sumter's blood tests "*remain negative for hemolysis*."[79] And finally, Sumter's hemoglobin reading at the time of his allegedly elevated plasma free hemoglobin reading was not only

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that test and using it all the time, we, you know, frequently see this kind of problem.").

[76] *See* Dirk Zimmerman's Medical Records, attached as Ex. 23.

[77] *See* Joe Sumter's Medical Records (7/11/01), attached as Ex. 24.

[78] *See* Dr. Banner's Expert Report, at 3, attached as Ex. 4; Depo. of Dr. Pike, at 98, ll. 1 – 16, attached as Ex. 14. *See also* D.S. Jacobs et al., LABORATORY TEST HANDBOOK (2nd ed. 1990).

[79] *Id.*

not below 10 g/dl (or even below normal, as one would expect in cases of arsine poisoning), but it was actually slightly ***above*** the laboratory normal range.[80]

The plaintiffs' experts, however, simply chose to ignore these facts and the questions they raise. For example, Dr. Harrison testified that he did not know whether Sumter's sample was lipemic,[81] did not know how plasma free hemoglobin is measured,[82] and did not know whether lipemia would interfere with the test.[83] He also testified repeatedly that Sumter's plasma free hemoglobin reading is evidence of hemolysis because it was "ten times normal,"[84] but he did not understand (*because he did not understand the units of measurement*) that, even at ten times normal, Sumter's plasma free hemoglobin reading was still 13 times lower than what he himself identified as being sufficient to "confirm[] the diagnosis of arsine poisoning."[85]

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80 *See* Chart of Clinical Findings for Twelve Plaintiffs, attached as Ex. 10. Likewise, Mr. Sumter's haptoglobin levels were normal, indicating that this reading was an artifact. *See* Dr. Banner's Expert Report, at 3, attached as Ex. 4.

81 *See* Depo. of Dr. Harrison, at 240, ll. 12-25, attached as Ex. 1 ("I did not see any results of their blood lipids.").

82 *Id.* at 238, l. 9-11 ("I don't know how the labs do it.").

83 *Id*. at 239, ll. 6-11 ("I don't know.").

84 *Id.* at 347, ll. 21-25; 428, ll. 9-14.

85 *See* Harrison, *supra* note 34, at 974.

II. The Novel Theories Offered By Dr. Hastings Cannot Show Injury To Plaintiff.

Dr. Hastings originally provided expert reports for all but a hand-full of the 192 plaintiffs that opined that the arsine released on July 11, 2001 caused them immediate and permanent injuries. In a few of these reports, Dr. Hastings also opined that arsine transformed into arsenic once it entered the plaintiffs' bodies.[86] Years into this lawsuit, Dr. Hastings for the first time disclosed a new opinion that the arsine transformed into arsenic ***before*** it even reached the plaintiffs.[87] Solkatronic's motion to strike the late-revealed opinions of Dr. Hastings remains pending.[88] In any event, Dr. Hastings' arsenic theories cannot be validated. He does not know how much arsine was transformed into arsenic or how much arsenic it would take to cause injury to the plaintiffs, and ignores the lack of any evidence of hemolysis.

A. Without Evidence of Hemolysis, Plaintiffs' Experts Cannot Validate Any Injury to Plaintiffs from the Incident.

Dr. Hastings' theory that the arsine transformed into inorganic arsenic after it entered the plaintiffs' bodies still does not account for the lack of certain biological effects that must be present.[89] Dr. Hastings relies on a group of scientists who hypothesized that arsine is transformed into inorganic arsenic within a person's body after an acute exposure.[90] The medical data and case studies used in this experiment show the same biological effects of

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[86] *See, e.g.*, Dr. Hastings' Expert Report on Plaintiff Theresa Haggard, at 3-4, attached as Ex. 12.

[87] *See* Depo. of Dr. Hastings, at 141, ll. 2-21, attached as Ex. 3.

[88] Magistrate Judge Cleary deferred any ruling on the Motion to this Court. *See* Mag. Judge Cleary's Order dated 10/12/05 (Docket #82). Solkatronic appealed its Motion to Strike Dr. Hastings to this Court. *See* Docket # 83.

[89] Solkatronic's experts, Dr. Pike and Dr. Carter, point out that other studies disagree with the conclusion made in the articles relied upon by Dr. Hastings. *See* Dr. Pike's Expert Report, at 18, attached as Ex. 5 (citing S.M. Healy et al, *Enzymatic Methylation of Arsenic Compounds v. Arsenite Methyltransferase Activity in Tissues of Mice*, 148 TOXIC. APPL. PHARMACOL. 65 (Jan. 1998)); Dr. Carter's Expert Report, at 6, attached as Ex. 6.

[90] Pietro Apostoli et al, *Metabolism of Arsenic After Acute Occupational Arsine Intoxication*, 52 J. TOXIC. & ENVTL. HEALTH 331, 333 (1997); P.J. Landrigan et al, *Occupational Exposure to Arsine: An*

hemolysis and the triad of symptoms as any other acute arsine exposure.[91] As even Dr. Hastings admits, the individual referenced in this study experienced hemolysis,[92] as well as "dark red urine" the morning after the exposure.[93] Even Dr. Hastings agrees that a "hemolytic anemia injury to the red blood cell" is the mechanism for arsine poisoning, and that he has seen no scientific or medical literature that supports an injury from arsine that has transformed into arsenic without hemolysis.[94] As discussed fully above, none of the plaintiffs have the biological markers of hemolysis, the triad of symptoms or the needed laboratory results.

B. No Evidence Exists to Show That Arsenic Could Have Caused Injury to Plaintiffs.

Dr. Hastings' new theory that arsine transforms into arsenic *before* it reached a person's body depends completely on whether enough arsenic was produced and carried by the wind off of Solkatronic's property to injure the plaintiffs. The only evidence that arsine was transformed into arsenic is the small amount of arsenic found near the ruptured arsine cylinder.[95] Soil samples around Solkatronic after the incident reveal that the inorganic arsenic levels in the soil were well within the normal range of arsenic naturally found in the ground before the incident.[96] Even if arsine had transformed into arsenic before entering a plaintiff's body, not enough arsenic could have been produced to cause the effects attributed

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Epidemiological Reappraisal of Current Standards, 8 SCAND. J. WORK & ENVTL. HEALTH 169 (1982).–

[91] Apostoli, *supra* note 84, at 333; Landrigan, *supra* note 84, at 169 (1982).–

[92] *See* Depo. of Dr. Hastings, at 134, l. 24 – 135, l. 5, attached as Ex. 3.

[93] Apostoli, *supra* note 110, at 333.

[94] *See* Depo. of Dr. Hastings, at 240, ll. 7-16 & 266, lns. 4-8, attached as Ex. 3. Dr. Hastings' opinion admittedly contradicts that of the Medical Management Guidelines for Arsine. *Id*. at 268, ll. 7-13.

[95] Dr. Hasting testified that the only support for this statement is a statement made in the expert report prepared by Dr. Pike, Solkatronic's expert. Dr. Pike noted that the investigation of the incident showed that there was some arsenic around the ruptured arsine cylinder after the incident.

[96] *Compare* the URS Report, dated 10/19/2001, attached as Ex. 20 (concluding there was no apparent impacts to soil from the July 11, 2001 arsine release) with the USGS Report, attached as Ex. 21.

by Dr. Hastings.[97]

Without a shred of evidence, Dr. Hastings opines that the ultimate byproduct of this mystery chemical transformation from arsine to arsenic is arsenic trioxide.[98] Arsenic trioxide is a completely separate toxin from arsine with a completely different mechanism of injury.[99] In fact, arsenic trioxide is a particulate, not a gas like arsine, and appears as a crystalline powder that resembles sugar.[100] There are **no** reports in witness accounts, either from the plaintiffs or emergency responders, or in any of the records that describe seeing a powder or any type of particulate. Further, direct contact with arsenic trioxide particulate causes skin and eye irritation.[101] Again, there is absolutely no evidence of complaints in any of the plaintiffs of skin and/or eye irritation.

The Medical Management Guidelines for Arsenic Trioxide states, "[a]rsenic trioxide dust is readily absorbed from the lungs, but inhaled quantities are usually insufficient to cause acute systemic toxicity . . . .[102] Most acute intoxications are from suicidal or homicidal ingestion."[103] In arsenic intoxication, the typical and usually seen method of poisoning is through ingestion not inhalation.[104]

No proof exists that any of the plaintiffs came into contact with arsenic trioxide, to say nothing of inhaling or ingesting a sufficient quantity to cause a toxic response. Dr. Hastings testified that he was unaware of any of the plaintiffs reporting they felt a particulate

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97 Agency for Toxic Substances & Disease Registry, *Medical Management Guidelines for Arsenic Trioxide* 1, (May 2, 2004), *available at* http://www.atsdr.cdc.gov/MHMI/mmg168.html. [hereinafter Medical Management Guidelines for Arsenic Trioxide]].

98 *See* Depo. of Dr. Hastings, at 150, l. 20 – 151, l. 2, attached as Ex. 3.

99 *Medical Management Guidelines for Arsenic Trioxide, supra* note 94, at 1.

100 *Id.*

101 *Id.*

102 *Id.*

103 *Id.*

matter, or that any of the plaintiffs saw or reported any powder or particulate matter at the time of the release.[105] He did not even research what form of arsenic he was referring to, ("I haven't got that researched out yet")[106] or how much arsenic he believes resulted from the release.[107]

Dr. Hastings' theory is flawed both medically and scientifically. Arsine is a gas; arsenic is a solid.[108] The mechanism for arsine toxicity is inhalation; the mechanism for arsenic toxicity is ingestion.[109] The target organ for arsine is the erythrocyte; the target organs for arsenic are the gastrointestinal tract, heart, brain and kidneys. Arsine is a non-irritating gas; arsenic causes acute skin, eye and upper respiratory tract irritation. As stated, there is no evidence that arsenic caused injury to the plaintiffs in this case**.**

CONCLUSION

The plaintiffs' experts ignore the settled science and medicine of arsine exposure. Instead, they rely on isolated (and usually subjective) symptoms to opine that the plaintiffs were immediately and permanently injured from the arsine release. They did not bother to find out if these symptoms were produced by other more likely causes, and they boldly contradict years of scientific study of arsine. The plaintiffs' experts opinions cannot be validated and lack scientific merit.

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104 *Id.*
105 *Id.* at 160, ll. 10-19.
106 *Id*. at 106, ll. 18-22.
107 *Id*. at 159, ll. 5-17.
108 *Medical Management Guidelines for Arsenic Trioxide, supra* note 94, at 1.
109 *Id*.